Northumbria Research Link

Hypovolemic shock in Adults (at a glance)

Introduction

This article introduces the reader to hypovolemic shock. It discusses the risk factors, aetiology, investigations, staging, complications, principles of management, education and training, for hypovolemic shock.

Shock

Shock is generally classified according to its cause. There are four main pathological mechanisms that can result in a state of shock (Vincent and De Backer, 2013; Straton, 2017), being: hypovolemia (loss of intravascular volume form internal or external fluid loss, cardiogenic (pump failure), obstruction (barriers to cardiac filling or circulatory flow); and distributive shock (dur to vaso-regulation and loss of vascular tone).

Shock is most commonly defined as 'the life-threatening failure of adequate oxygen delivery to the tissues and may be due to decreased blood perfusion of tissues, inadequate blood oxygen saturation, or increased oxygen demand from the tissues that results in decreased end-organ oxygenation and dysfunction' (Stratton, 2019). If left untreated, shock results in sustained multiple organ dysfunction, and end-organ damage with possible death. Tissue hypoperfusion may be present without systemic

hypotension, but at the bedside shock is commonly diagnosed when both are present (arterial hypotension and organ dysfunction) (Stratton, 2019).

Hypovolemic Shock

Hypovolaemic shock is characterised by a loss of intavascular volume of 15% or more leading to inadequate perfusion of the tissues (Peate, 2020). Hypovolemic shock occurs when the volume of the circulatory system is too depleted to allow adequate circulation to the tissues of the body (Rull and Bonsall, 2017). In summary, hypo means low, vol means volume and anaemic means blood translating as low blood volume. Patients with hypovolemic shock have severe hypovolemia with decreased peripheral perfusion. If left untreated, these patients can develop ischemic injury of vital organs, leading to multi-system organ failure. The first factor to be considered is whether the hypovolemic shock has resulted from haemorrhage or fluid losses, as this will dictate treatment.

Pathophysiology and symptoms

Hypovolemic shock results from depletion of intravascular volume, either by blood loss or extracellular fluid loss. The body compensates for this with increased sympathetic tone resulting in increased heart rate and cardiac contractility, and peripheral vasoconstriction. Changes in vital signs include; increase in diastolic blood pressure with narrowed pulse pressure. As volume status decreases, systolic blood pressure drops. Oxygen delivery to vital organs is unable to meet oxygen demand as a result and cells switch from aerobic metabolism to anaerobic metabolism, resulting in lactic acidosis. Blood flow is diverted from other organs to

preserve blood flow to the heart and brain as sympathetic drive increases. This propagates tissue ischemia and worsens lactic acidosis. If this is not corrected, there will be worsening hemodynamic compromise and, eventually, death (Gayet_Ageron et al., 2018)

Symptoms of hypovolemic shock can be related to volume depletion, electrolyte imbalances or acid base disorders that accompany hypovolemic shock. Patients with volume depletion may experience thirst, muscle cramps and/or orthostatic hypotension (decrease in systolic blood pressure of 20mmHG or decrease in diastolic blood pressure of 10mmHG within three minutes of standing compared to a sitting or supine blood pressure). In severe hypovolemic shock patients can experience abdominal or chest pain caused by mesenteric and coronary ischemia. Brain malperfusion can cause agitation, lethargy or confusion. Physical assessments as a result of volume depletion may find dry mucous membranes, decreased skin elasticity, low jugular venous distention. Tachycardia, hypotension and decreased urinary output. Patients can also appear cold, clammy and cyanotic (Annane et al., 2013).

Risk factors

A healthy adult can withstand the loss of half a litre from a circulation of about five litres without ill effect (Rull and Bonsall, 2017); however, larger volumes and rapid loss cause progressively greater problems. Risk is very much related to the degree of hypovolaemia and the speed of correction. In children and young adults' tachycardia is one of the earliest signs of hypovolaemia as the circulatory system is much better able to cope with the rigours of loss. The risk of morbidity and mortality

is much greater as age increases because they often do not tolerate having low blood volume (Rull and Bonsall, 2017). Pathology in the cardiovascular, respiratory and renal systems increases risk.

Aetiology

The annual incidence of shock of any aetiology is 0.3 to 0.7 per 1000, with haemorrhagic shock being most common in the intensive care unit (Taghavi and Askari, 2019). Hypovolemic shock is the most common type of shock in children, most commonly due to diarrheal illness in the developing world. Hypovolemic shock occurs as a result of either blood loss or extracellular fluid loss. Haemorrhagic shock is hypovolemic shock from blood loss. Traumatic injury is by far the most common cause of haemorrhagic shock. Other causes of haemorrhagic shock include gastrointestinal (GI) bleed, bleed from an ectopic pregnancy, bleeding from surgical intervention, or vaginal bleeding (Taghavi and Askari, 2019).

Hypovolemic shock as a result of extracellular fluid loss can be of the following aetiologies:

Gastrointestinal Losses

GI losses can occur via many different aetiologies. The gastrointestinal tract usually secretes between 3 to 6 litres of fluid per day. However, most of this fluid is reabsorbed as only 100 to 200 mL are lost in the stool. Volume depletion occurs when the fluid ordinarily secreted by the GI tract cannot be reabsorbed. This occurs

when there is retractable vomiting, diarrhoea, or external drainage via stoma or fistulas. (Taghavi and Askari, 2019).

Renal Losses

Renal losses of salt and fluid can lead to hypovolemic shock. The kidneys usually excrete sodium and water in a manner that matches intake. Diuretic therapy and osmotic diuresis from hyperglycaemia can lead to excessive renal sodium and volume loss. In addition, there are several tubular and interstitial diseases beyond the scope of this article that cause severe salt-wasting nephropathy (Taghavi and Askari, 2019).

Skin Losses

Fluid loss also can occur from the skin. In a hot and dry climate, skin fluid losses can be as high as 1 to 2 litres/hour. Patients with a skin barrier interrupted by burns or other skin lesions also can experience large fluid losses that lead to hypovolemic shock (Taghavi and Askari, 2019).

Third-Space Sequestration

Sequestration of fluid into a third-space also can lead to volume loss and hypovolemic shock. Third-spacing of fluid can occur in intestinal obstruction, pancreatitis, obstruction of a major venous system, or any other pathological condition that results in a massive inflammatory response (Taghavi and Askari, 2019).

Blood Work

Monitoring electrolytes and acid/base status in patients in hypovolemic shock is of utmost importance. Biochemical analysis will identify any electrolyte and acid-base

disturbances for example contraction alkalosis, metabolic acidosis which could affect choice of replacement fluid and rate of repletion. In some cases arterial blood gas is needed if mixed acid-base disturbance is suspected (Galvagno, 2014)

Multiple organ dysfunction syndrome (MODS) (Procter, 2019)

The combination of direct and reperfusion injury may cause MODS—the progressive dysfunction of ≥ 2 organs consequent to life-threatening illness or injury (Procter, 2019). MODS can follow any type of shock but is most common when infection is involved; organ failure is one of the defining features of septic shock. MODS also occurs in > 10% of patients with severe traumatic injury and is the primary cause of death in those surviving > 24 hours (Procter, 2019).

Any organ system can be affected, but the most frequent target organ is the lung, in which increased membrane permeability leads to flooding of alveoli and further inflammation. Lactate production and metabolic acidosis produces abnormal levels of hydrogen ions. This decreases the PH in the blood and the level of bicarbonate. In order to compensate, respiratory rate is increased resulting in hyperventilation (Galvagno, 2014). Progressive hypoxia may be increasingly resistant to supplemental oxygen therapy. This condition is termed acute lung injury or, if severe, acute respiratory distress syndrome (ARDS) (Procter, 2019).

The kidneys are injured when renal perfusion is critically reduced, leading to acute tubular necrosis and renal insufficiency manifested by oliguria and progressive rise in serum creatinine (Procter, 2019).

In the heart, reduced coronary perfusion and increased mediators (including tumor necrosis factor and interleukin-1) may depress contractility, worsen myocardial compliance, and down-regulate beta-receptors. These factors decrease cardiac output, further worsening both myocardial and systemic perfusion and causing a vicious circle often culminating in death. Arrhythmias may occur what ones? (Procter, 2019).

Aby Mitchell 1/4/2020 14:27
Comment [1]: Barry, I can't find this.

In the gastrointestinal tract, ileus and submucosal hemorrhage can develop. Liver hypoperfusion can cause focal or extensive hepatocellular necrosis, transaminase and bilirubin elevation, and decreased production of clotting factors (Procter, 2019).

Coagulation can be impaired, including the most severe manifestation, disseminated intravascular coagulopathy (Procter, 2019).

Investigations (Rull and Bonsall, 2017)

Investigation	**Rationale**
Check Haemaglobin (Hb), Urea and Electrolytes (U&E), Liver Function Test (LFT) and, in haemorrhage and burns, group and save and crossmatch	There is likely to be a significant drop in Hb in early stages of shock. Prompt administration of blood is essential in instances of severe or

	ongoing blood loss.
Coagulation screen	
Blood gases – arterial blood gas (ABG) or venous blood gas (VBG)	These may show a metabolic acidaemia from poor perfusion; lactate levels particularly reflect hypoperfusion. **Note:** In clinical practice, an ABG is always preferred as the respiratory component is captured, and with patients who are in shock, it is inevitable that they will deteriorate.
Monitor urine output, which may require a catheter.	Urine output should be used to guide administration of fluids.
Ultrasound	This can be useful for differentiating hypovolaemic from cardiogenic shock; the vena cava can be assessed for adequate filling and echocardiogram can show any pump failure.
Central venous pressure (CVP)	Monitoring CVP may be useful where there is evidence of shock.

Aby Mitchell 1/4/2020 14:34
Comment [2]: Barry can you add the rationale for this?

Staging

Lavoie, (2018) recognises that there are four stages of hypovolemic shock based on how much blood volume has been lost. All stages require early treatment, but it is helpful to recognise the stage of hypovolemia a person is in, so they receive appropriate treatment quickly.

Stage 1

During the earliest stage of hypovolemic shock, a person with will have lost up to 15 percent, or 750 ml, of their blood volume. This stage can be difficult to diagnose. Blood pressure, urine output and breathing will still be normal. The most noticeable symptom at this stage is skin that appears pale. The person may also experience sudden anxiety.

Stage 2

In the second stage, the body has lost up to 30 percent, or 1500 ml, of blood. The individual may experience increased heart and breathing rates. Blood pressure may still be within normal range. However, the diastolic pressure, or bottom number, of their blood pressure may be high. The person may begin sweating and feeling more anxious and restless. Capillary refill is delayed. Urine output of about 20-30 millilitres/hour.

Stage 3

By stage 3, a person with hypovolemic shock will have 30 to 40 percent, or 1500 to 2000 ml, blood loss. The top number or systolic pressure of their blood pressure will be 100 mm Hg or lower. Their heart rate will increase to over 120 beats per minute (bpm). They will also have a rapid breathing rate of over 30 breaths per minute. They

will begin to experience mental distress, including anxiety and agitation. The skin will be pale and cold, and they will begin sweating. Urine output drops to 20 millilitres/hour. Altered mental state will present in confusion, anxiety and/or agitation.

Stage 4

A person with stage-4 hypovolemia faces a critical situation. They will have experienced a loss of blood volume greater than 40 percent, or 2,000 ml. They will have a weak pulse but extremely rapid heart rate. Breathing will become be very fast and difficult. Systolic blood pressure will be under 70 millimetres of mercury (mm/Hg). They may experience the following symptoms:

1. drifting in and out of consciousness
2. sweating heavily
3. feeling cool to the touch
4. looking extremely pale
5. Absent capillary refill
6. Negligible urine output

Principle of Managing Hypovolemic Shock

Management of hypovolemia involves assessing and treating the underlying cause, identifying electrolyte and acid-base disturbances, and assessing and treating the volume deficit, all of which influence the choice of fluid and rate at which it should be administered (Mandel and Palevsky, 2019).

Clinicians should identify the aetiology (or aetiologies) contributing to hypovolemia so that therapies can be directed at the underlying cause of volume loss. Potential aetiologies of hypovolemia include gastrointestinal, renal, skin, haemorrhage, and third-space losses. Therapies may include anti-emetics to treat vomiting, cessation of diuretics, or controlling bleeding. Further details regarding aetiology and diagnosis of hypovolemia are discussed separately.

Identify electrolyte and acid-base disturbances — Biochemical analysis will alert the clinician to electrolyte (e.g., hypo- or hypernatremia, hypo- or hyperkalaemia) and acid-base disturbances (e.g., contraction alkalosis, metabolic acidosis) which may affect choice of replacement fluid and rate of repletion. In some cases, an arterial blood gas may be needed if mixed acid-base disturbance is suspected.

Fluid Resuscitation

It is suggested that fluid resuscitation should be commenced immediately to restore circulating volume and improve cardiac output. NICE (2017) advise that crystalloids are most appropriate, unless the patient presents with active internal or external bleeding. In such cases, Red Blood Cells should be transfused as the patient would require haemoglobin to support the transportation around the body to prevent anaerobic respiration and cell death causing increase lactate (Dutton and Finch, 2018). Highlight carriage problems earlier and that respiratory rate and SaO2 may indicate this issue.

Aby Mitchell 1/4/2020 15:01
Comment [3]: Barry, I can't answer this

Use of Crystalloids (NICE, 2017)

NICE (2017) recommend the administration of intravenous (IV) crystalloids that contain sodium in the range 130–154 mmol/l, with a bolus of 500 ml over less than 15 minutes. (For more information, see the Composition of commonly used crystalloids - table 1.)

Composition of commonly used crystalloids

Content	Plasma	Sodium chloride 0.9%*	Sodium chloride 0.18%/ 4% glucose[a]	0.45% NaCl/ 4% glucose[a]	5% glucose[a]	Hartmann's	Lactated Ringer's (USP)	Ringer's acetate	Alternative balanced solutions for resuscitation**	Alternative balanced solutions for maintenance**
Na^+ (mmol/l)	135–145	154	31	77	0	131	130	130	140	40
Cl^- (mmol/l)	95–105	154	31	77	0	111	109	112	98	40
$[Na^+]$:$[Cl^-]$ ratio	1.28–1.45:1	1:1	1:1	1:1	-	1.18:1	1.19:1	1.16:1	1.43:1	1:1
K^+ (mmol/l)	3.5–5.3	*	*	*	*	5	4	5	5	13
HCO_3^- / Bicarbonate	24–32	0	0	0	0	29 (lactate)	28 (lactate)	27 (acetate)	27 (acetate) 23 (gluconate)	16 (acetate)
Ca^{2+} (mmol/l)	2.2–2.6	0	0	0	0	2	1.4	1	0	0
Mg^{2+} (mmol/l)	0.8–1.2	0		0		0	0	1	1.5	1.5
Glucose (mmol/ l)	3.5–5.5	0	222 (40 g)	222 (40 g)	278 (50 g)	0	0	0	0	222 (40 g)
pH	7.35–7.45	4.5–7.0	4.5		3.5–5.5	5.0–7.0	6–7.5	6–8	4.0–8.0	4.5–7.0
Osmolarity (mOsm/l)	275–295	308	284		278	278	273	276	295	389

* These solutions are available with differing quantities of potassium already added, and the potassium-containing versions are usually more appropriate for meeting maintenance needs.
** Alternative balanced solutions are available commercially under different brand names and composition may vary by preparation.
[a] The term dextrose refers to the dextro-rotatory isomer of glucose that can be metabolised and is the only form used in IV fluids. However IV fluid bags are often labelled as glucose so only this term should be used. Traditionally hospitals bought a small range of fluids combining saline (0.18-0.9%) with glucose but several recent NICE/NPSA documents have recommended specific combinations, which are now purchased to enable guidelines to be followed. Glucose–saline combinations now come in 5 different concentrations, and the addition of variable potassium content expands the pre-mixed range to 13 different products. Prescribers must therefore specify the concentration of each component; the term dextrose-saline (or abbreviation D/S) is meaningless without these details. What is specified also impacts significantly on the cost of the product.
Note: Weight-based potassium prescriptions should be rounded to the nearest common fluids available (for example, a 67 kg person should have fluids containing 20 mmol and 40 mmol of potassium in a 24-hour period). Potassium should not be added to intravenous fluid bags as this is dangerous.

Source: This table was drafted based on the consensus decision of the members of the Guideline Development Group.

'Intravenous fluid therapy in adults in hospital', NICE clinical guideline 174 (December 2013. Last update December 2016)

Table 1: Composition of commonly used crystalloids (NICE, 2017) Editor – Please redraw.

Complications (Rull and Bonsall, 2017)

Complication	**Wider implication**
Blood is directed away from the kidneys and gut	This can produce acute kidney injury and complications of gut ischaemia
Obstetric shock	Acute tubular necrosis can occur

Inadequate perfusion	This leads to hypoxia and metabolic acidosis
About 75% of the blood flow to the right ventricle and 100% to the left ventricle occurs in diastole	A fall in diastolic pressure will predispose to cardiac arrhythmias and even arrest. Upset of acid-base balance, hypoxia and disturbance of electrolytes will aggravate the problem
In those who are susceptible, dehydration	This may lead to haemoconcentration and sludging of the circulation with such complications as venous sinus thrombosis.

Vasoactive drugs

Following the infusion of either crystalloids (fluid loss) or RBC's (after blood loss), the patients vital signs should be remeasured. It is imperative that the patient has a normotensive blood pressure to perfuse the brain, heart, lungs and kidneys particularly, as otherwise they are likely to develop an Acute Kidney Injury or go into shock (REF). This is the goal of fluid resuscitation and has no mention of vasoactive drugs.

Consider the audience for this paper. These drugs are most often administered in critical care areas.

Aby Mitchell 1/4/2020 14:38
Comment [4]: Barry, do we still need this?

Pearls and Other Issues (Taghavi and Askari, 2019)

In patients with hypovolemic shock due to extracellular fluid loss, the aetiology of fluid loss must be identified and treated.

Monitoring electrolytes and acid/base status in patients in hypovolemic shock is of utmost importance.

Trauma is the leading cause of haemorrhagic shock.

The haemorrhagic shock should be treated with balanced transfusion of packed red blood cells, plasma, and platelets.

Determining whether patients will be responsive to volume resuscitation should not rely on a single modality such as ultrasound, pulse pressure wave variation, passive leg raises, or central venous pressure. The decision for fluid administration should be based on a complete systematic assessment to help direct volume resuscitation.
For patients with hypovolemic shock due to fluid loss, the crystalloid solution is preferred over colloid.

Training and education (NICE, 2017)

Hospitals should establish systems to ensure that all healthcare professionals involved in prescribing and delivering IV fluid therapy are trained on the principles covered in this guideline, and are then formally assessed and reassessed at regular intervals. Health care professionals need to be able to assess, identify and escalate care. Competence must be demonstrated in:

- understanding the physiology of fluid and electrolyte balance in patients with normal physiology and during illness

- assessing patients' fluid and electrolyte needs (the 5 Rs: Resuscitation, Routine maintenance, Replacement, Redistribution and Reassessment)
- assessing the risks, benefits and harms of IV fluids
- prescribing and administering IV fluids
- monitoring the patient response
- evaluating and documenting changes and
- taking appropriate action as required.
- Hospitals should have an IV fluids lead, responsible for training, clinical governance, audit and review of IV fluid prescribing and patient outcomes.

Conclusion

Hypovolemic shock is characterised by an imbalance between oxygen supply and demand. If left untreated patients can develop ischemic injury of vital organs, which leads to multi-system organ failure. It is important for nurses to be able to assess, identify and escalate care to ensure patients receive correct and timely treatment.

Reference List

Annane D, Siami S, Jaber S, Martin C, Elatrous S, Declère AD, Preiser JC, Outin H, Troché G, Charpentier C, Trouillet JL, Kimmoun A, Forceville X, Darmon M, Lesur O, Reignier J, Régnier J, Abroug F, Berger P, Clec'h C, Cle'h C, Cousson J, Thibault L, Chevret S., CRISTAL Investigators. Effects of fluid resuscitation with colloids vs crystalloids on mortality in critically ill patients presenting with hypovolemic shock: the CRISTAL randomized trial. JAMA. 2013 Nov 06;310(17):1809-17.

Dutton, H. Finch, J. (2018) Acute and Critical Care Nursing at a Glance. Wiley Blackwell publishers.

Galvagno, S., 2014. *Emergency Pathophysiology*. Hoboken: Teton NewMedia, Inc.

Gayet-Ageron A, Prieto-Merino D, Ker K, Shakur H, Ageron FX, Roberts I., Antifibrinolytic Trials Collaboration. Effect of treatment delay on the effectiveness and safety of antifibrinolytics in acute severe haemorrhage: a meta-analysis of individual patient-level data from 40 138 bleeding patients. Lancet. 2018 Jan 13;391(10116):125-132.

Hooper N, Armstrong TJ. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): May 6, 2019. Haemorrhagic Shock.

Lavoie, L. (2018) What to know about hypovolemic shock. Medical News Today. Available at: https://www.medicalnewstoday.com/articles/312348.php (Last Accessed: 24.01.2020).

Mandel, J. Palevsky, P. (2019) Treatment of severe hypovolemia or hypovolemic shock in adults. Available at: https://www.uptodate.com/contents/treatment-of-severe-hypovolemia-or-hypovolemic-shock-in-adults (accessed on: 15.01.2020).

NICE (2017) Intravenous Fluid therapy in Adult in Hospital. NICE Clinical Guideline 174. Available at: https://www.nice.org.uk/guidance/cg174/resources/composition-of-commonly-used-crystalloids-table-191662813 (Last Accessed 24.01.2020).

Procter, L. (2019) Shock. Available at: https://www.msdmanuals.com/en-gb/professional/critical-care-medicine/shock-and-fluid-resuscitation/shock (accessed: 16.01.2020).

Rull, G. Bonsall, A. (2017) Resuscitation in Hypovolaemic Shock. Emergency medicine and trauma. Available at: https://patient.info/doctor/resuscitation-in-hypovolaemic-shock (Accessed on: 16.01.2020).

Stratton, S. (2019) Shock. BMJ Best Practice. Available at: https://bestpractice.bmj.com/topics/en-gb/1013#referencePop4 (Accessed on: 15.01.202).

Taghavi, S. Askari, R. (2019) *Hypovolemic shock*. Available at: https://www.ncbi.nlm.nih.gov/books/NBK513297/ (Accessed on: 15.01.2020).

Vincent, J. De Backer, De. (2013) Circulatory Shock. *NEJM.* 2013: 369 (18); 1726-1734.